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DATE: Monday, September 27, 2004

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<input type="checkbox"/>	L4	L1 near5 (promoter or regulat? region or cis\$ element or "5 UTR")	2
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<input type="checkbox"/>	L2	L1 and (promoter or regulat? region or cis\$ element or "5' UTR")	162
<input type="checkbox"/>	L1	growth differentiation factor 9 or GDF-9	204

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=> s growth differentiation factor 9 or GDF 9
L1 359 GROWTH DIFFERENTIATION FACTOR 9 OR GDF 9

=> s l1 and (promoter or regulat? region? or regulat? element? or 5 UTR)
L2 11 L1 AND (PROMOTER OR REGULAT? REGION? OR REGULAT?
ELEMENT? OR 5
UTR)

=> dup rem l2
PROCESSING COMPLETED FOR L2

L3 6 DUP REM L2 (5 DUPLICATES REMOVED)

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L3 ANSWER 1 OF 6 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS
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on STN DUPLICATE 1
AN 2003342043 EMBASE
TI GCNF-dependent repression of BMP-15 and ***GDF*** - ***g*** mediates
gamete regulation of female fertility.
AU Lan Z.-J.; Gu P.; Xu X.; Jackson K.J.; DeMayo F.J.; O'Malley B.W.; Cooney
A.J.
CS A.J. Cooney, Dept. of Molec. and Cellular Biology, Baylor College of
Medicine, One Baylor Plaza, Houston, TX 77030, United States.
a.cooney@bcm.tmc.edu
SO EMBO Journal, (15 Aug 2003) 22/16 (4070-4081).
Refs: 48
ISSN: 0261-4189 CODEN: EMJODG
CY United Kingdom
DT Journal; Article
FS 010 Obstetrics and Gynecology
029 Clinical Biochemistry
LA English
SL English
AB To determine the function of germ cell nuclear factor (GCNF) in female
reproduction, we generated an oocyte-specific GCNF knockout mouse model
(GCNF(f/f))Zp3Cre(+)). These mice displayed hypofertility due to
prolonged diestrus phase of the estrous cycle and aberrant
steroidogenesis. These reproductive defects were secondary to a primary
defect in the oocytes, in which expression of the paracrine transforming
growth factor- β , signaling molecules, bone morphogenetic protein 15
(BMP-15) and ***growth*** ***differentiation*** ***factor***
g (***GDF*** - ***g***), were up-regulated in GCNF
(f/f)Zp3Cre(+) females at diestrus. This was a direct effect of GCNF, as
molecular studies showed that GCNF bound to DR0 elements within the BMP-
15
and ***GDF*** - ***g*** gene promoters and repressed their reporter
activities. Consistent with these findings, abnormal double-oocyte
follicles, indicative of aberrant BMP-15 ***GDF*** - ***g***
expression, were observed in GCNF (f/f)Zp3Cre(+) females. The Cre/loxP
knockout of GCNF in the oocyte has uncovered a new regulatory pathway in
ovarian function. Our results show that GCNF directly regulates paracrine
communication between the oocyte and somatic cells by regulating the
expression of BMP-15 and ***GDF*** - ***g***, to affect female
fertility.

L3 ANSWER 2 OF 6 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation.
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DUPLICATE 2
AN 2003:40151 BIOSIS
DN PREV200300040151
TI ***Growth*** ***differentiation*** ***factor*** - ***g***
stimulates inhibin production and activates Smad2 in cultured rat
granulosa cells.
AU Roh, Jae-Sook; Bondestam, Jonas; Mazerbourg, Sabine; Kaivo-oja, Noora;
Groome, Nigel; Ritvos, Olli; Hsueh, Aaron J. W. [Reprint Author]
CS Department of Gynecology and Obstetrics, Stanford University School of
Medicine, Stanford, CA, 94305-5317, USA
aaron.hsueh@stanford.edu
SO Endocrinology, (January 2003) Vol. 144, No. 1, pp. 172-178. print
CODEN: ENDOAO. ISSN: 0013-7227.
DT Article
LA English
ED Entered STN: 15 Jan 2003
Last Updated on STN: 15 Jan 2003
AB Ovarian inhibin production is stimulated by FSH and several TGFbeta family
ligands including activins and bone morphogenetic proteins.
Growth ***differentiation*** ***factor*** - ***g*** (***GDF*** - ***g***)
derived by the oocyte is a member of the
TGFbeta/activin family, and we have previously shown that ***GDF*** - ***g***
treatment stimulates ovarian inhibin-alpha content in explants
of neonatal ovaries. However, little is known about ***GDF*** - ***g***
regulation of inhibin production in granulosa cells and
downstream signaling proteins activated by ***GDF*** - ***g***.
Here, we used cultured rat granulosa cells to examine the influence of
GDF - ***g*** on basal and FSH-stimulated inhibin production,
expression of inhibin subunit transcripts, and the ***GDF*** - ***g***
activation of Smad phosphorylation. Granulosa cells from small antral
follicles of diethylstilbestrol-primed immature rats were cultured with
FSH in the presence or absence of increasing concentrations of ***GDF***
- ***g***. Secreted dimeric inhibin A and inhibin B were quantified
using specific ELISAs, whereas inhibin subunit RNAs were analyzed by
Northern blotting using 32P-labeled inhibin subunit cDNA probes. Similar
to FSH, treatment with ***GDF*** - ***g*** stimulated dose- and
time-dependent increases of both inhibin A and inhibin B production.
Furthermore, cocultivation of cells with ***GDF*** - ***g*** and FSH
led to a synergistic stimulation of both inhibin A and inhibin B
production. ***GDF*** - ***g*** treatment also increased mRNA
expression for inhibin-alpha and inhibin-beta subunits. To investigate
Smad activation, granulosa cell lysates were analyzed in immunoblots using
antiphosphoSmad1 and antiphosphoSmad2 antibodies. ***GDF*** - ***g***
treatment increased Smad2, but not Smad1, phosphorylation with increasing

doses of ***GDF*** - ***g*** leading to a dose-dependent increase in phosphoSmad2 levels. To further investigate inhibin-alpha gene ***promoter*** activation by ***GDF*** - ***g***, granulosa cells were transiently transfected with an inhibin-alpha ***promoter***-luciferase reporter construct and cultured with different hormones before assaying for luciferase activity. Treatment with FSH or ***GDF*** - ***g*** resulted in increased inhibin-alpha gene ***promoter*** activity, and combined treatment with both led to synergistic increases. The present data demonstrate that oocyte-derived ***GDF*** - ***g***, alone or together with pituitary-derived FSH, stimulates inhibin production, inhibin subunit mRNA expression, and inhibin-alpha ***promoter*** activity by rat granulosa cells. The synergistic stimulation of inhibin secretion by the paracrine hormone ***GDF*** - ***g*** and the endocrine hormone FSH could play an important role in the feedback regulation of FSH release, thus leading to the modulation of follicle maturation and ovulation.

L3 ANSWER 3 OF 6 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation.
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DUPLICATE 3

AN 2002:401575 BIOSIS

DN PREV200200401575

TI ***Growth*** ***differentiation*** ***factor*** - ***g***

inhibits 3'-adenosine monophosphate-stimulated steroidogenesis in human granulosa and theca cells.

AU Yamamoto, Noriko; Christenson, Lane K.; McAllister, Jan M.; Strauss, Jerome F., III [Reprint author]

CS Center for Research on Reproduction and Women's Health, 421 Curie Boulevard, 1354 Biomedical Research Building II/III, Philadelphia, PA, 19104, USA
jfs3@mail.med.upenn.edu

SO Journal of Clinical Endocrinology and Metabolism, (June, 2002) Vol. 87, No. 6, pp. 2849-2856, print.
CODEN: JCEMAZ. ISSN: 0021-972X.

DT Article

LA English

ED Entered STN: 24 Jul 2002

Last Updated on STN: 29 Aug 2002

AB ***Growth*** ***differentiation*** ***factor*** - ***g*** (***GDF*** - ***g***), a member of the transforming growth factor superfamily, modulates the development and function of granulosa and theca cells. Targeted deletion of ***GDF*** - ***g*** in the mouse revealed that ***GDF*** - ***g*** was essential for the establishment of the thecal cell layer during early folliculogenesis. During later stages of follicular development, the roles of ***GDF*** - ***g*** are less well understood, but it has been postulated that oocyte-derived ***GDF*** - ***g*** may prevent premature luteinization of follicular cells, based on its ability to modulate steroidogenesis by rodent ovarian cells. In the rodent, ***GDF*** - ***g*** is expressed solely by the oocyte from the early primary follicular stage through ovulation. Recent studies in the rhesus monkey demonstrated that granulosa cells express ***GDF*** - ***g***, suggesting a broader role for this protein in ovarian function in primates. We examined the effect of recombinant ***GDF*** - ***g*** on proliferating human granulosa and thecal cell steroidogenesis and the expression of steroidogenic acute regulatory protein (StAR), P450 side-chain cleavage, and P450 aromatase. We also examined granulosa cell ***GDF*** - ***g*** expression by quantitative RT-PCR and by Western analysis. ***GDF*** - ***g*** inhibited 8-Br-cAMP-stimulated granulosa progesterone synthesis by approximately 40%, but did not affect basal progesterone production. Concordant with reduced steroid production, 8-Br-cAMP-stimulated StAR protein expression was reduced approximately 40% in granulosa cells, as were expression of StAR mRNA and StAR ***promoter*** activity. Additionally, ***GDF*** - ***g*** inhibited 8-Br-cAMP-stimulated expression of P450 side-chain cleavage and P450 aromatase. Human granulosa cells expressed ***GDF*** - ***g***, as determined by RT-PCR and Western analysis. Treatment of human thecal cells with ***GDF*** - ***g*** blocked forskolin-stimulated progesterone, 17alpha-hydroxyprogesterone, and dehydroepiandrosterone synthesis. Thecal cells exhibited greater sensitivity to ***GDF*** - ***g***, suggesting that this cell may be a primary target of ***GDF*** - ***g***. Moreover, ***GDF*** - ***g*** increased thecal cell numbers during culture, but had no effect on granulosa cell growth. Our findings implicate ***GDF*** - ***g*** in the modulation of follicular steroidogenesis, especially theca cell function. Because ***GDF*** - ***g*** mRNA and protein are detectable in granulosa-lutein cells after the LH surge, the concept of ***GDF*** - ***g*** as a solely oocyte-derived luteinization inhibitor needs to be reevaluated.

L3 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1999:640978 CAPLUS

DN 131:267961

TI Transcription regulatory sequences derived from mouse ***growth***

differentiation ***factor*** - ***g*** (***GDF*** - ***g***) gene and methods to modulate tissue-specific expression

IN Matzuk, Martin Matthew; Elvin, Julia Andrea

PA Metamorphix, Inc., USA

SO PCT Int. Appl., 37 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9950406	A2	19991007	WO 1999-US7185	19990331
WO 9950406	A3	19991118		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GV, ML, MR, NE, SN, TD, TG				
CA 2324286	AA	19991007	CA 1999-2324286	19990331
AU 9934626	A1	19991018	AU 1999-34626	19990331
EP 1070134	A2	20010124	EP 1999-916271	19990331
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 9909288	A	20011127	BR 1999-9288	19990331
JP 2004512003	T2	20040422	JP 2000-541294	19990331
PRAI US 1998-80108P	P	19980401		
WO 1999-US7185	W	19990331		
AB Isolated ***GDF*** - ***g*** regulatory sequences are disclosed, as well as methods of using the sequences to modulate tissue-specific expression of genes. The ***GDF*** - ***g*** regulatory sequences include, for example, enhancer and ***promoter*** elements that naturally drive transcription of ***GDF*** - ***g*** in specific tissues. The ***GDF*** - ***g*** regulatory sequences can be derived from the untranscribed upstream (e.g., first 10 kilobases) and downstream regions, and transcribed, untranslated regions of a ***GDF*** - ***g*** gene. Marked ***GDF*** - ***g*** mRNA accumulation was shown both in the ovary and the testis of transgenic mice contg. ***GDF*** - ***g*** -regulatory sequence constructs.				

L3 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1999:487225 CAPLUS

DN 131:120903

TI Methods and compositions for enhancing cognitive function using morphogenetic proteins

IN Charette, Marc F.

PA Creative Biomolecules, Inc., USA

SO PCT Int. Appl., 74 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9937320	A1	19990729	WO 1999-US1232	19990121
W: AU, CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 2003170213	A1	20030911	US 1998-12846	19980123
AU 9923309	A1	19990809	AU 1999-23309	19990121
EP 1047443	A1	20001102	EP 1999-903241	19990121
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
EP 1398039	A1	20040317	EP 2003-23804	19990121
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
US 2004176292	A1	20040909	US 2003-734472	20031212
PRAI US 1998-12846	A	19980123		
EP 1999-903241	A3	19990121		
WO 1999-US1232	W	19990121		
AB Disclosed are methods and compns. for protecting cognitive function in a mammal, particularly a human, subject to brain tissue damage, by administering a morphogen or a nucleic acid encoding a morphogen to the mammal. The methods and compns. can be used to reduce memory dysfunction and/or to provide a neuroprotective effect in subjects at risk of memory dysfunction resulting from a mech. or chem. trauma, neuropathies, neurodegenerative diseases, or oxygen or glucose deprivation.				
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD				
ALL CITATIONS AVAILABLE IN THE RE FORMAT				

L3 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1999:375567 CAPLUS

DN 131:28319

TI Maintenance of vascular smooth muscle integrity by morphogenic proteins

IN Nakaoka, Takashi; Miyazono, Kohei; Sampath, Kuber T.

PA Creative Biomolecules, Inc., USA

SO PCT Int. Appl., 41 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9928341	A2	19990610	WO 1998-US25398	19981130
WO 9928341	A3	19990805		
W: AU, CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,				

PT, SE
CA 2314423 AA 19990610 CA 1998-2314423 19981130
AU 9917064 A1 19990616 AU 1999-17064 19981130
EP 1037910 A2 20000927 EP 1998-961838 19981130
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, FI
PRAI US 1997-67690P P 19971204
WO 1998-US25398 W 19981130

AB Disclosed are compns. and methods for maintaining the integrity of smooth muscle, particularly vascular smooth muscle. Vascular diseases are characterized by an excessive build-up of vascular smooth muscle cells, resulting in an occlusion of a blood vessel, and/or by loss of elasticity in the blood vessels. Causes of blood vessel occlusion include smooth muscle cell proliferation and inflammatory responses. Inhibition of the proliferation of smooth muscle cells or inflammatory responses represents an effective treatment for vascular disorders, such as atherosclerosis and restenosis. Treatment may include administration of a morphogenic protein. The protein itself may be delivered to the site of vascular occlusion or the protein may be delivered by a vector, such as an adenoviral vector contg. a DNA insert encoding a morphogenic protein. Such compns. and methods may also inhibit the responses of smooth muscle cells to various traumas, such as exposure to toxic agents. All of these treatments operate to preserve the cell phenotype by inhibiting an increase in extracellular matrix proteins, such as collagen, or by maintaining the normal balance of extracellular matrix proteins, such as Types I and III collagen.

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L2 11 S L1 AND (PROMOTER OR REGULAT? REGION? OR REGULAT? ELEMENT? OR
L3 6 DUP REM L2 (5 DUPLICATES REMOVED)

=> s l1 and (mouse or murine)
L4 152 L1 AND (MOUSE OR MURINE)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 88 DUP REM L4 (64 DUPLICATES REMOVED)

=> s l5 and (promoter or regulat? region? or regulat? element? or 5 UTR)
L6 2 L5 AND (PROMOTER OR REGULAT? REGION? OR REGULAT? ELEMENT? OR 5 UTR)

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L6 ANSWER 1 OF 2 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation.
on STN
AN 2002:401575 BIOSIS
DN PREV200200401575
TI ***Growth*** ***differentiation*** ***factor*** - ***g***
inhibits 3'-adenosine monophosphate-stimulated steroidogenesis in human granulosa and theca cells.
AU Yamamoto, Noriko; Christenson, Lane K.; McAllister, Jan M.; Strauss, Jerome F., III [Reprint author]
CS Center for Research on Reproduction and Women's Health, 421 Curie Boulevard, 1354 Biomedical Research Building II/III, Philadelphia, PA, 19104, USA
jfs3@mail.med.upenn.edu
SO Journal of Clinical Endocrinology and Metabolism, (June, 2002) Vol. 87, No. 6, pp. 2849-2856. print.
CODEN: JCEMAZ. ISSN: 0021-972X.
DT Article
LA English
ED Entered STN: 24 Jul 2002
Last Updated on STN: 29 Aug 2002

AB ***Growth*** ***differentiation*** ***factor*** - ***g*** (***GDF*** - ***g***), a member of the transforming growth factor superfamily, modulates the development and function of granulosa and theca cells. Targeted deletion of ***GDF*** - ***g*** in the ***mouse*** revealed that ***GDF*** - ***g*** was essential for the establishment of the thecal cell layer during early folliculogenesis. During later stages of follicular development, the roles of ***GDF*** - ***g*** are less well understood, but it has been postulated that oocyte-derived ***GDF*** - ***g*** may prevent premature luteinization of follicular cells, based on its ability to modulate steroidogenesis by rodent ovarian cells. In the rodent, ***GDF*** - ***g*** is expressed solely by the oocyte from the early primary follicular stage through ovulation. Recent studies in the rhesus monkey demonstrated that granulosa cells express ***GDF*** - ***g***, suggesting a broader role for this protein in ovarian function in primates. We examined the effect of recombinant ***GDF*** - ***g*** on proliferating human granulosa and thecal cell steroidogenesis and the expression of steroidogenic acute regulatory protein (StAR), P450 side-chain cleavage, and P450 aromatase. We also examined granulosa cell ***GDF*** - ***g*** expression by quantitative RT-PCR and by Western

analysis. ***GDF*** - ***g*** inhibited 8-Br-cAMP-stimulated granulosa progesterone synthesis by approximately 40%, but did not affect basal progesterone production. Concordant with reduced steroid production, 8-Br-cAMP-stimulated StAR protein expression was reduced approximately 40% in granulosa cells, as were expression of StAR mRNA and StAR ***promoter*** activity. Additionally, ***GDF*** - ***g*** inhibited 8-Br-cAMP-stimulated expression of P450 side-chain cleavage and P450 aromatase. Human granulosa cells expressed ***GDF*** - ***g***, as determined by RT-PCR and Western analysis. Treatment of human thecal cells with ***GDF*** - ***g*** blocked forskolin-stimulated progesterone, 17alpha-hydroxyprogesterone, and dehydroepiandrosterone synthesis. Thecal cells exhibited greater sensitivity to ***GDF*** - ***g***, suggesting that this cell may be a primary target of ***GDF*** - ***g***. Moreover, ***GDF*** - ***g*** increased thecal cell numbers during culture, but had no effect on granulosa cell growth. Our findings implicate ***GDF*** - ***g*** in the modulation of follicular steroidogenesis, especially theca cell function. Because ***GDF*** - ***g*** mRNA and protein are detectable in granulosa-lutein cells after the LH surge, the concept of ***GDF*** - ***g*** as a solely oocyte-derived luteinization inhibitor needs to be reevaluated.

L6 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:640978 CAPLUS
DN 131:267961
TI Transcription regulatory sequences derived from ***mouse***
growth ***differentiation*** ***factor*** - ***g*** (***GDF*** - ***g***) gene and methods to modulate tissue-specific expression
IN Matzuk, Martin Matthew; Elvin, Julia Andrea
PA Metamorphix, Inc., USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9950406	A2	19991007	WO 1999-US7185	19990331
WO 9950406	A3	19991118		
W:	AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
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CA 2324286	AA	19991007	CA 1999-2324286	19990331
AU 9934626	A1	19991018	AU 1999-34626	19990331
EP 1070134	A2	20010124	EP 1999-916271	19990331
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO			
BR 9909288	A	20011127	BR 1999-9288	19990331
JP 2004512003	T2	20040422	JP 2000-541294	19990331
PRAI US 1998-80108P	P	19980401		
WO 1999-US7185	W	19990331		

AB Isolated ***GDF*** - ***g*** regulatory sequences are disclosed, as well as methods of using the sequences to modulate tissue-specific expression of genes. The ***GDF*** - ***g*** regulatory sequences include, for example, enhancer and ***promoter*** elements that naturally drive transcription of ***GDF*** - ***g*** in specific tissues. The ***GDF*** - ***g*** regulatory sequences can be derived from the untranscribed upstream (e.g., first 10 kilobases) and downstream regions, and transcribed, untranslated regions of a ***GDF*** - ***g*** gene. Marked ***GDF*** - ***g*** mRNA accumulation was shown both in the ovary and the testis of transgenic mice contg. ***GDF*** - ***g***-regulatory sequence constructs.

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